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Bilateral neurological deficits following unilateral minimally invasive TLIF: A review of four patients

Alexander T. Nixon, Zachary A. Smith, Cort D. Lawton, Albert P. Wong, Nader S. Dahdaleh, Antoun Koht[1], Richard G. Fessler[2]

Departments of Neurological Surgery and [1]Anesthesiology, Department of Neurological Surgery, Department of Neurology, Northwestern University, Chicago, IL 60661, [2]Department of Neurological Surgery, Rush University Neurosurgery, Chicago, IL 60612, USA

E-mail: Alexander T. Nixon - alexander.nixon@my.rfums.org; Zachary A. Smith - zsmithmd@gmail.com; Cort D. Lawton - cort.lawton@my.rfums.org; Albert P. Wong - a-wong@fsm.northwestern.edu; Nader S. Dahdaleh - nsdahdaleh@gmail.com; Antoun Koht - a-koht@northwestern.edu; *Richard G. Fessler - rfessler@rush.edu

*Corresponding author

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Abstract

Background: Minimally invasive transforaminal lumbar interbody fusion (MI-TLIF) is commonly used for the treatment of degenerative lumbar spinal disorders. The rate of postoperative neurological deficits is traditionally low. New neurological postoperative complications may be underreported. We report our infrequent rate of MI-TLIF procedures complicated by postoperative weakness.

Methods: A database of 340 patients was evaluated, all of whom underwent MI-TLIF procedures performed between January 2002 and June 2012 by the senior author. We identified four cases (1.2%) whose postoperative course was complicated with bilateral lower extremity weakness. We retrospectively reviewed their past medical history, operative time, estimated blood loss, length of hospital stay, changes in intraoperative neurophysiological monitoring, and pre- and postoperative neurological exams.

Results: The average age of the four patients was 65.5 years(range: 62-75 years), average body mass index (BMI) was 25.1 (range: 24.1-26.6), and there were three females and one male. All patients had preoperative degenerative spondylolisthesis (either grade I or grade II). All patients were placed on a Wilson frame during surgery and underwent unilateral left-sided MI-TLIF. Three out of the four patients had a past medical history significant for abdominal or pelvic surgery and one patient had factor V Leiden deficiency syndrome.

Conclusions: The rate of new neurological deficits following an MI-TLIF procedure is low, as documented in this study where the rate was 1.2%. Nonetheless, acknowledgement and open discussion of this serious complication is important for surgeon education. Of interest, the specific etiology or pathophysiology behind these complications remains relatively unknown (e.g. direct neural injury, traction injury, hypoperfusion, positioning complication, and others) despite there being some similarities between the patients and their perioperative courses.

Key Words: Complications, lumbar, minimally invasive, spine, transforaminal lumbar interbody fusion

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INTRODUCTION

Advances in intraoperative imaging and surgical instrumentation have led to the development of the minimally invasive transforaminal lumbar interbody fusion (MI-TLIF) approach.[10,21] The transforaminal lumbar interbody fusion (TLIF) approach introduced by Harms and Rolinger allows for interbody fusion and foraminal decompression via a unilateral dorsal approach. It may be preferred by some surgeons because of the potential for decreased thecal sac and nerve root retraction, and preservation of the contralateral tissue when compared to the posterior lumbar interbody fusion (PLIF).[8]

The complication rates reported comparing MI-TLIF and open counterparts vary between studies.[15,18,23] Neurological complications are usually unilateral and are related to direct neural injury due to misplaced hardware, dissection, and retraction. Studies report neurological complication rates from 0 to 10.96%, with an average rate of 5.76%.[18,23] In this study, new postoperative neurological complications encountered by a single surgeon were reviewed for 340 consecutive patients undergoing MI-TLIF. This manuscript reports the details of a complication not previously reported from the unilateral MI-TLIF approach. Possible etiologies that might have contributed to this rare complication are discussed.

MATERIALS AND METHODS

A database of 340 consecutively treated MI-TLIF patients was created using Current Procedural Terminology (CPT) codes and the senior author's records from January 2002 to June 2012. Inclusion criteria included single-level and multi-level MI-TLIFs, and virgin or revision surgeries. Common diagnoses included spondylolisthesis, degenerative disk disease, and spondylosis [Table 1]. Patients were not included in the database if the procedure involved an open approach or was part of a long-construct scoliosis revision. Additionally, cases of trauma or infection were excluded. The majority of cases were single-level (87.65%) MI-TLIFs, predominantly at L4–L5 (54.41%) followed by the L5-S1 (26.47%) level.

Institutional review board (IRB) approval was obtained from Northwestern University and University of Chicago. Patient records were reviewed for demographics, past medical history, clinical and radiographic findings, intraoperative notes, anesthesia flow charts, progress notes, and any consults occurring in the postoperative period prior to discharge. Neurophysiologic monitoring report-dictations of somatosensory evoked potentials (SSEPs), electromyograms (EMGs), and motor evoked potentials (MEPs), as well as the exact intraoperative tracings recorded throughout each case were reviewed.

Surgical technique

A single senior surgeon performed surgeries, with the patient under general anesthesia in the prone position on a Wilson frame. The MI-TLIF technique is described elsewhere.[14] Preoperative and postoperative neurological exams were complete, including lower extremity sensation, reflexes, and muscle strength. Muscle strength was graded on the standard 5-point scale.

RESULTS

Incidence of new postoperative neurological deficits

The incidence of new postoperative neurological deficits was 1.2%. These patients averaged 65.5 years of age

Table 1: Demographic and clinical data for the entire series of 340 consecutive MI-TLIF patients

Parameter	*n* of patients or mean±SD	% of patients
Number	340	100
Mean age±SD (years)	60±13.26	
Gender (female)	181	53.23
Preoperative diagnosis		
Spondylosis	13	3.82
Spondylolisthesis	209	61.47
Prior failed surgery	11	3.23
Adjacent segment disease	3	0.88
Spondylolysis	9	2.65
Degenerative disk disease	140	41.18
Recurrent disk herniation	4	1.18
Level of fusion		
L1-L2	1	0.29
L2-L3	4	1.18
L2-L4	4	1.18
L2-L5	1	0.29
L3-L4	17	5
L3-L5	15	4.41
L3-S1	1	0.29
L4-L5	185	54.41
L4-S1	21	6.18
L5-L6	1	0.29
L5-S1	90	26.47
Single-level fusion	298	87.65
Multiple-level fusion	42	12.35
BMP use	329	96.76
Perioperative data		
Operative time (min)	215.36±72.01	
Blood loss (ml)	159.82±137.06	
Length of stay (days)	3.46±2.19	

BMP: Bone morphogenic protein, SD: Standard deviation, MI-TLIF: Minimally invasive transforaminal lumbar interbody fusion

Table 2: Patient demographics and known past medical and surgical histories

Case no.	Age (years)	Sex	BMI (kg/m^2)	Past medical history	Past surgical history
1	63	Male	24.1	Melanoma Osteoarthritis Atrial fibrillation Hypertension Benign prostatic hyperplasia Pulmonary congestion	Radical groin dissection
2	62	Female	25.7	DVT×2 Multiple PEs AAA (3.5 cm) Hypertension Hyperlipidemia Anxiety Factor V Leiden	L4-L5 laminectomy Right hip replacement Breast reduction Hysterectomy
3	62	Female	24.2	Anxiety	Bilateral Lasik eye surgery NSVD×2
4	75	Female	26.6	Hypercholesterolemia Glaucoma Left renal cancer Colon cancer	Left elbow surgery Hysterectomy Left nephrectomy Colectomy SVT ablation

AAA: Abdominal aortic aneurysm, DVT: Deep vein thrombosis, PE: Pulmonary embolism, NSVD: Normal spontaneous vaginal delivery, SVT: Supraventricular tachycardia

Table 3: Operative procedures and perioperative parameters

Case no.	Operation	Cage size	Cage removal	OR time (min)	EBL	Perioperative complications
1	Left L4-L5 TLIF	10×25 at L4-L5	No	141	50	A-fib w/RVR
2	Left L4-L5 TLIF Left L5-S1 TLIF Vascular: IVCF	9×25 at L4-L5; 6×25 at L5-S1	No	357	100	None
3	Left L4-L5 TLIF	8×25 at L4-L5	Yes	187	200	None
4	Left L4-L5 TLIF	10×25 at L4-L5	No	186	50	None

EBL: Estimated blood loss, IVCF: Inferior vena cava filter, OR: Operating room, RVR: Rapid ventricular response, TLIF: Transforaminal lumbar interbody fusion

(range: 62-75 years) and had average body mass indexes (BMIs) of 25.1 (range: 24.1-26.6). There were three females and one male. Three of the four patients had a documented history of previous abdominal or pelvic procedures [Table 2]. All patients had presented with preoperative lower back pain and radiculopathy involving the L5 dermatome. All patients had normal, baseline muscle strength established preoperatively, except for patient #2 who presented with dorsiflexion weakness (4/5) in the left lower extremity. Three patients had grade I spondylolisthesis at the L4-L5 level. Patient #2 had grade II spondylolisthesis at L4-L5 and grade I spondylolisthesis at L5-S1. All four patients were treated with a unilateral, left-sided, MI-TLIF. Three patients had single-level procedures at L4-L5. Patient #2 had a two-level procedure at L4-L5 and L5-S1. Cage size details are presented in Table 3. The mean operative time was 218 min (range: 141-357 min) and the average estimated blood loss (EBL) was 100 ml (range: 50-200 ml). Patient #1 experienced an additional perioperative complication of atrial fibrillation with rapid ventricular response. The overall complication rate of the database was below 10%; there were four patients (1.2%) with new postoperative bilateral foot drop.

Pathological changes in intraoperative monitoring

Four patients had intraoperative decreases or loss of SSEPs [Table 4]. Patient #1 had loss of SSEPs during soft tissue dissection. Because of this patient's extreme instability, the decision was made to continue with placement of an interbody cage and posterior instrumentation. Neuromonitoring in patient #2 revealed complete loss of the SSEP signals in the left lower extremity after the placement of the cage, while the right lower extremity showed a decrease in amplitude. This patient underwent a wake-up test intraoperatively and the motor test revealed movement in right leg with left leg at baseline. So, the decision was made to continue with the procedure. Patient #3 had bilateral loss of SSEPs in the lower extremities after insertion of the cage, with subsequent return of SSEPs after cage removal. So, the cage was removed and bone morphogenic protein was placed deeply into the disk space. Patient #4 had MEP changes after inserting the trials, followed by decreased SSEP amplitudes. Complete loss of SSEPs in patient #4 was observed 16 min after initial MEP changes followed by partial recovery; changes in right lower extremity were not as severe. Because of the recovery, the decision was made to proceed with a smaller cage placement. An overview of each patient's neuromonitoring changes is shown in Table 4, and Figure 1 reveals the waterfall tracings for patient #2 to patient #4. No abnormal spontaneous EMG discharges were observed with any of the four patients.

Postoperative neurological examination: Foot drop

Patients #1 and #2 both had more significant postoperative lower extremity weakness than patients #3 and #4. Both plantar flexion and dorsiflexion were lost bilaterally in patient #1 immediately postoperatively; these movements partially returned in the left lower extremity at discharge, 6 days later. Clinically, 1 year later, patient #1 returned to full strength in the left lower extremity, but had residual deficits (1/5) in both plantar flexion and dorsiflexion of the right lower extremity. Patient #2 also had loss of dorsiflexion and plantar flexion immediately postoperatively, with minimal return on discharge, 5 days

later. Some improvement was seen in clinic at 9 months, with dorsiflexion and plantar flexion being noted as 3/5 in both bilateral lower extremities. Patient #3 had full plantar flexion strength postoperatively, but had limited dorsiflexion and extensor hallucis longus function (right lower extremity worse than left). Patient #3 returned to full (baseline) muscle strength by 1-year follow-up. Patient #4 had clinical weakness (3-4/5) with dorsiflexion and plantar flexion in both lower extremities postoperatively and at discharge. Her function returned clinically at 1 year, except for her left extensor hallucis longus muscle. There were no changes between pre- and postoperative bladder or sphincter tone with any of the four patients. Average hospital stay was 4.3 days (range: 2-6 days), and all patients were discharged to an acute inpatient rehabilitation facility.

Table 4: Neuromonitoring changes

Case no.	Change in neuromonitoring	Time of change	Neuromonitoring progression
1	Acute decrease BLE SSEPs	Soft tissue dissection	SSEPs slowly improved during surgery No abnormal spontaneous EMG discharges observed
2	>90% Decrease in BLE SSEPs	After cage placement	Wake-up test: RLE moved well, LLE mild weakness Slight improvement in RLE SSEP, but not LLE at closing No abnormal spontaneous EMG discharges observed
3	Loss of BLE SSEPs	After cage placement	SSEPs slowly improved after cage removal No abnormal spontaneous EMG discharges observed
4	Loss of BLE SSEPs and MEPs	10 mm cage trial	MEPs began returning bilaterally and cage was inserted Right SSEP present with diminished amplitude at closing No abnormal spontaneous EMG discharges observed

BLE: Bilateral lower extremity, EMG: Electromyogram, LLE: Left lower extremity, MEP: Motor evoked potentials, RLE: Right lower extremity, SSEP: Somatosensory evoked potential

The computed tomography (CT) and magnetic resonance images (MRI) images shown in Figures 2 and 3 are examples of the postoperative imaging findings in the patients discussed in this report. There was no evidence of neural compression in any patient postoperatively.

DISCUSSION

Infrequent complications of MI-TLIF are largely underreported

Minimally invasive (MI) procedures involve smaller operating fields, require more radiological imaging, and are more technically challenging.[15,18] Complications associated with MI-TLIF have been well documented, but there is a paucity of studies addressing new neurological deficits. Thus, it is possible that this rare complication is largely underreported. Honest appraisal of significant deficits is critical to understand the risks of commonly performed operations. This manuscript discusses this infrequent but significant complication.

Overall complication rate for MI-TLIF

The overall complication rate for MI-TLIF procedures has been reported as 0-40%.[2,13] Few studies report neurological complications other than radiculopathies, which are often transient. New neurological complications such as weakness, or foot drop, are rarely reported. We identified new postoperative weakness in 4/340 (1.2%) patients. The injury pattern and distribution of the neurological deficits was not uniform, which is not alarming considering the etiology, or the mechanism of injury is unknown. Rosen *et al.* and Wu *et al.* each reported weakness in a single patient [0.9% and

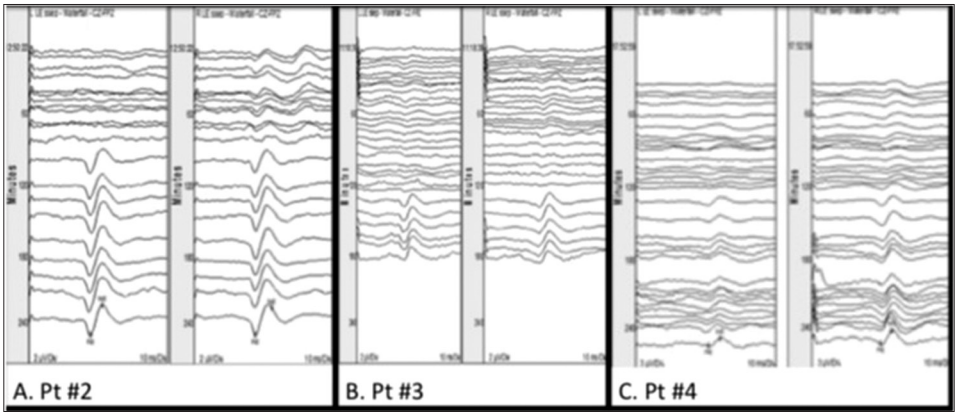

Figure 1: (a) Patient #2. The tracing from the left lower extremity shows a complete loss of the SSEP signals after the placement of the cage, while the right lower extremity shows a decrease in the amplitude. (b) Patient #3. There was a bilateral loss from the lower extremities seen after the insertion of the cage. Signals were partially recovered after the removal of the cage. (c) Patient #4. Left lower extremity SSEPs decreased in amplitude shortly after MEPs, followed by a complete loss of SSEPs 16 min later. Changes in right lower extremity were not as severe and showed some recovery

Figure 2: Postoperative imaging of the illustrative case, patient #4 demonstrating cage placement. CT imaging lateral (a) and axial (c). X-ray imaging lateral (b) and AP (d)

0.66% (1/107 and 1/151), respectively] postoperatively after an MI-TLIF.[20,25] Villavicencio *et al.* divided the complications into major and minor, depending on the duration of the neurological deficit (more or less than 3 months). In an earlier study, they reported neurological deficits in 8/73 (11%) patients (3 major and 5 minor), and in a more recent study, the neurological complication rate was reported as 10.5% (8/76) (5 major and 3 minor).[22,23] In a study of 108,419 patients in which the rates of new neurological deficits associated with spine surgery were assessed, Hamilton *et al.* reported an overall rate of approximately 1%, with higher rates found in cases of fusion or implants.[7]

Neurophysiological monitoring changes with MI-TLIF interbody cage placement

In a recent study, Duncan *et al.* described neurophysiological monitoring change secondary to interbody cage placement in five patients undergoing a TLIF; two patients developed new postoperative neurological findings.[6] The cases involved reversal of SSEPs back to baseline post-removal of the interbody cage; so they suggested that distraction of the nerve root caused stretching of the nerve and vascular supply associated with the nerve root. The study by Duncan *et al.* examines patients following TLIF with a midline incision, whereas our patients were treated with an MI approach. Furthermore, the two patients with newly developed deficits consisted of one with cauda equina dysfunction and another one with unilateral weakness of the left foot. Each of our patients presented with bilateral weakness and had foot drop. In patient #3, SSEP amplitude partially improved following cage removal, which may indicate stretch was partially implicated.

Etiology of stretch injury

When the distance a nerve traverses increases, it is prone to stretch injury; failure of nerve conduction has been

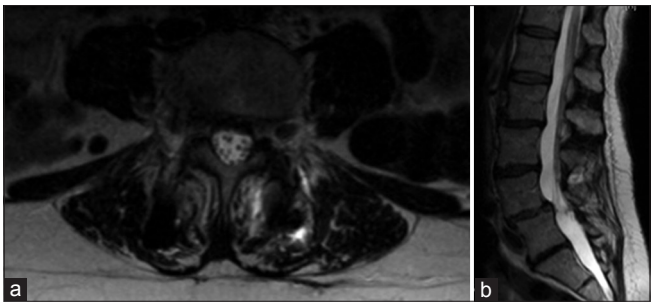

Figure 3: Postoperative imaging of patient #3. MRI imaging axial (a) and sagittal (b)

reported with stretch levels 6% or above.[5,11] Kitab *et al.* reviewed the anatomic basis for injury at the lumbar spine and suggested that nerve stretch may be the primary source of pathological pain and/or injury.[11] Risk associated with stretch injuries is not linear; nerves are more prone to stretch injuries occurring as the reduction approaches completion. The majority (71%) of total nerve strain occurs in the final half of slip reduction.[19] Stretch injury would be more likely in high-grade (3-5) spondylolisthesis; the four patients discussed in the current manuscript had low-grade spondylolisthesis. Vascular hypoperfusion and ischemia to the peripheral nerve may parallel structural changes incurred during a stretch injury. Ischemia can result from a 15% elongation with histological manifestations noted between 4 and 50% elongation.[11]

Spinal perfusion considerations with intraoperative monitoring

Spinal perfusion is intimately involved with neurophysiological functioning. A report by Jayson *et al.* suggests a role for vascular damage and fibrosis in nerve root injury.[9] Disk degeneration can compress epidural veins, dilating uncompressed epidural veins. The vascular changes can lead to venous obstruction, thromboses, perineural fibrosis, hypoxia, and nerve root damage with subsequent neuronal atrophy.[9] In addition, caudal regions of the spinal cord heavily rely on the great radicular artery (artery of Adamkiewicz or arteria radicularis magna), which is typically found at L1 or L2 on the left.[17] All the cases presented in this study were an MI-TLIF from a left-sided approach. Lo *et al.* reported an anatomical variant of the artery of Adamkiewicz in which the artery's origin is higher; they found three cases in which the fourth lumbar artery flowed into the ASA of the conus medullaris.[16]

Vaculopathy attributed to hypercoagulable states

When discussing perfusion, it is of interest to note that at least one patient had factor V deficiency and other patients might have been "vasculopaths" with an underlying "hypercoaguable state," considering their history of cancer. The true implications of the patient's comorbidities and the previous surgeries relative to spinal perfusion are unknown.

Contributing factors which may predispose to foot drop with MI-TLIF

One potential exacerbating factor may be the Wilson frame used for operative positioning. A recent study examined the risk factors for developing postoperative ischemic optic neuropathy and found the use of the Wilson frame to be independently associated with ischemic optic neuropathy.[1] The suggested pathophysiology was increased intra-abdominal pressure. The role of vascular perfusion was also noted by Bhardwaj *et al.*, who reported neurological deficits after laminectomies in which the patient was in a prone position.[3] In this regard, it is interesting to note that patient #1 in this series reported preoperatively that following sleeping prone, he experienced severe pain and weakness in his lower extremities.

A second potential exacerbating factor could stem from the observation that three of the four patients in our series reported a history of previous abdominal or pelvic surgery [Table 2]. Insult to the peritoneum, or mesothelial damage, can lead to intra-abdominal scarring or adhesions; damage may be the result of surgery, trauma, or inflammation.[4] Two female patients had a history of hysterectomy and one of them had an additional nephrectomy and colectomy for cancer. One male patient had a history of radical groin dissection. Furthermore, adhesions may be inflammatory as reported in a postmortem examination by Weibel *et al.* that reported 28% adhesions in patients without previous surgery.[24]

One patient did not have a documented history of previous abdominal surgery, but did have a history of Leiden factor V deficiency and, therefore, had a baseline hypercoagulable state. These observations led us to consider the possibility that previous abdominal surgery and/or hypercoagulable states could alter lumbar spinal perfusion.

Importance of intraoperative neurophysiological monitoring to spine surgery

Intraoperative neurophysiological monitoring is an important adjunct tool to the neurosurgeon. All the four cases included in the current report had changes in SSEPs. With all patients, a drop in SSEPs was followed by thorough exploration of the wound and dura, in addition to checking the five potential causes of SSEP changes: Technical, physiological, pharmacological, positional, and surgical. The first three patients who had SSEP changes were monitored by SSEPs and EMGs. The last patient had surgery after we implemented the use of MEP monitoring, in addition to SSEPs and EMGs. The abrupt bilateral signal SSEP/MEP loss in the lower extremities without changes in lower extremity EMG (or upper extremities) suggests stretch or vascular rather than mechanical cause for such changes. None of the patients recorded abnormal spontaneous EMG discharges; this is consistent with an ischemic insult, as EMG monitors mechanical or thermal injuries.

There were three other patients in the larger database with intraoperative neuromonitoring changes. Only one patient was symptomatic, presenting with unilateral left lower extremity weakness. The symptoms were transient and resolved within a month of discharge. Multiple modalities are used in conjunction for neuromonitoring as the sensitivities, specificities, and advantages/disadvantages vary with each monitoring technique.[12]

CONCLUSION

MI-TLIF is commonly employed for the treatment of degenerative lumbar disorders. There are numerous common, minor complications and relatively infrequent, serious complications. Complications may be underreported, especially serious complications such as postoperative neurological deficits or weakness. We identified 4 cases (1.2%) of bilateral weakness after a unilateral MI-TLIF. No definitive conclusions can be drawn about the specific risk factors from the present study. However, factors which predispose to hypoperfusion, such as previous abdominal surgery, hypercoagulability, and increased intra-abdominal pressure, suggest interesting contributory possibilities. Future work should be directed at addressing these possibilities.

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Commentary

The authors of the study entitled "Bilateral neurological deficits following unilateral minimally invasive transforaminal lumbar interbody fusion (MI-TLIF): A review of four patients" should be congratulated for brining up a major issue – the need to report new neurological deficits resulting from spinal surgery. Here, the authors retrospectively evaluated the frequency of new neurological deficits developing in 340 patients undergoing MI-TLIF (2002-2012); they found four (1.2%) patients with new bilateral foot drops. In their study, they evaluated what factors may have predisposed patients to these complications: Past medical history, operative time, estimated blood loss, length of hospital stay, changes in intraoperative neurophysiological monitoring, and pre- and postoperative neurologic exams. Their patients averaged 65.5 years of age (range: 62-75 years), had an average BMI of 25.1 (range: 24.1-26.6), and included three females and one male. They all exhibited a preoperative grade I or grade II degenerative spondylolisthesis and all underwent unilateral left-sided MI-TLIF.

VARIOUS ETIOLOGIES FOR NEUROLOGICAL COMPLICATIONS

Although multiple factors may have contributed to four new postoperative foot drops in these patients, the most probable cause was retraction/stretch/distraction injuries likely occurring during the placement of interbody cages. Other etiologies may have included vascular/ischemic compromise, operative positioning (e.g. Wilson frame in this study), vasculopathy, a variant of the artery of Adamkiewicz, prior abdominal surgery/adhesions, or coagulopathy (e.g. factor V Leiden in one patient this study).

LITERATURE DOCUMENTED

Overall complication rate is 0-40%. This includes all complications such as more common minor complications (durotomy, etc).[1,3] Two authors specifically discussed single neurological complications occurring in their MI-TLIF series involving 1 of 107 (0.96%) and 1 of 151 (0.6%) patients, respectively.[4,7] Another author, in two sequential MI-TLIF studies, observed a higher incidence of major and minor neurological complications: 11% (3 major and 5 minor of 73) in 2006 and 10.5% (5 major and 3 minor of 76) in 2010.[5,6]

SSEP CHANGES SIGNALING NEUROLOGICAL INJURY

In this study, significant intraoperative somatosensory evoked potential (SSEP) deterioration signaled the onset of new foot drops in all four patients. In five of Duncan *et al*.'s patients undergoing TLIF, intraoperative SSEP changes warned of the onset of new neural damage occurring during interbody cage placement. Although all changes reversed intraoperatively, two patients exhibited new postoperative neurological deficits.[2]

ARE INTRAOPERATIVE SSEP CHANGES BEING CAREFULLY MONITORED WITH TLIF?

Why do not we hear more about the frequency of SSEP changes/loss occurring during TLIF performed open or minimally invasively? Is SSEP monitoring presumably being utilized throughout these procedures,

not just during screw placement (e.g. along with EMG)? Furthermore, when many SSEP changes occur, they often demonstrate slow progressive deterioration rather than abrupt/complete drop-out; this "early warning" often provides some opportunity for resuscitative maneuvers (e.g. cessation of traction, removal of the interbody device to avoid over-distraction, etc.) to avoid permanent neurological deficits/sequelae.

NEW NEUROLOGIC POSTOPERATIVE COMPLICATIONS (OF MI-TLIF) ARE LARGELY UNDERREPORTED

The authors of the study are to be congratulated for acknowledging that new postoperative neurological deficits arise during MI-TLIF, and are largely "underreported". Indeed, the authors found a 1.2% incidence of new foot drops in 4 of their 340 patients. Furthermore, only a handful of other authors quoted the frequencies of new postoperative neurological deficits as varying from 0.6 to 11%.

STARTING A COMPLICATIONS CORNER FOR SNI SPINE SUPPLEMENT?

Should we more accurately inform our patients and peers regarding the incidence of adverse events/risks of open or MI-TLIF along with other spinal procedures? The data are there, but the means to report/publish this information has not been readily available. We will, therefore, start a Complications Corner to be published as part of the *Surgical Neurology International: Spine Supplement*. We look forward to your participation.

Nancy E. Epstein

Winthrop University Hospital, Chief of Neurosurgical Spine, Education, and Research, Long Island, NY, USA
E-mail: nancy.epstienmd@gmail.com

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